

Cardiac Gated ASL: Effects of CBF Pulsatility on the ASL Perfusion Signal

D. S. Bolar^{1,2}, T. Benner², E. Adalsteinsson^{1,2}, and A. G. Sorensen^{1,2}

¹Harvard-MIT Division of Health Sciences and Technology, Cambridge, MA, United States, ²MGH/HMS/MIT Martinos Center for Biomedical Imaging, Charlestown, MA, United States

Introduction

Arterial spin labeling (ASL) MRI has become a standard technique for measuring cerebral blood flow (CBF). Typical ASL experiments are based on the standard model for quantitative perfusion imaging [1], which assumes uniform plug blood flow. As a result, acquisition methods do not consider the cardiac cycle and have a fixed TR. By cardiac gating the ASL experiment, it is possible to exploit CBF pulsatility and increase the perfusion signal. In this study, we evaluate the effects of cardiac gating on ASL perfusion-SNR, and provide theoretical and experimental evidence suggesting that by optimizing the delay between the ECG trigger and ASL module, perfusion-SNR can be maximized.

Methods

Two human subjects were imaged on a Siemens 3T system, equipped with a 12-channel receive coil. Pulsed ASL experiments using a PICORE/QUIPSSII tagging scheme and EPI readout were performed [2]. The pulse sequence was modified to trigger off the QRS complex of the ECG signal. The timing diagram is visualized in figure 1a; a new parameter, T_{ECG} introduces a delay between the trigger and start of the ASL module. ASL imaging parameters were $TI_{PRE} = 700$ ms, $TI_{POST} = 700$ ms, inversion band thickness = 150 mm, ASL gap = 10 mm, TR for non-gated acquisition = 2500 ms, TR for gated acquisition was variable (but >2500 ms), FOV = 220 mm, 64x64 matrix, 72 measurements, slice thickness = 5 mm, interslice gap = 1 mm. For the ECG gated scans, T_{ECG} ranged between 0 ms and 1000 ms, in increments of 100 ms. Six axial slices were positioned to intersect the cerebral cortex. An IR-EPI sequence, with identical slice parameters, was run ($TI = 525$ ms) to effectively null white matter. The resulting images were thresholded to create gray matter (GM) masks.

Perfusion time-series data were generated by performing pairwise subtractions between tag and control images. SNR maps were subsequently created by averaging individual voxel values across the time series, and dividing by the standard deviation. The SNR maps were overlaid with the GM masks and mean SNR within the mask was calculated. This mean SNR was plotted against T_{ECG} . All analysis was performed with NeuroLens software [3].

Theory

Perfusion signal in ASL is dependent on the volume of tagged spins delivered to the imaging slab. Spin delivery is directly related to 1) flow during the interval between tag creation and tag saturation (TI_{PRE}), and 2) flow during the subsequent interval between saturation and imaging commencement (TI_{POST}). The first interval defines the maximum volume of tagged spins that can be delivered to the imaging slab. This volume is equal to the amount of blood that leaves the inversion band during TI_{PRE} (i.e. before saturation), and is proportional to the area under the flow profile bound by the interval. By the end of the first interval, the volume of deliverable tagged spins is fixed. Flow during the second interval subsequently controls the extent of delivery. During this interval, tagged spins continue to disseminate through the vascular tree towards and within the destination tissue. Thus, the total volume of tagged spins delivered depends on the flow profiles bounded by both intervals.

Introduction of the T_{ECG} parameter allows the two intervals to coincide with different segments of the flow profile. Graphically, this can be seen in figures 1b and 1c, which contain archetypal carotid/vertebral artery flow waveforms, extrapolated from ref [4]. Figure 1b depicts a scenario where ASL perfusion signal is maximal. Inversion occurs at the beginning of the high flow segment. The volume of deliverable tagged spins (red shaded region) is maximized, as is the proportion of these spins that will eventually reach the destination tissue (blue shaded region). Figure 1c repeats the experiment with a different T_{ECG} . In this case, the red shaded region is minimized, indicating that deliverable tagged spin volume is small. Despite high flow during the second interval (large blue region), the final perfusion signal is ultimately limited by this small deliverable volume, resulting in suboptimal perfusion-SNR. A third possible scenario (not shown) is if T_{ECG} is chosen such that the blue shaded region is minimized. Despite a large deliverable spin volume, the lack of flow in the second interval limits the extent of delivery, also resulting in suboptimal perfusion-SNR. These examples highlight the idea that signal in the final perfusion map relies heavily on the position of the ASL module in relation to the flow profile, and therefore on choice of T_{ECG} . Without ECG triggering, the position of the ASL module will fluctuate from measurement to measurement, yielding suboptimal perfusion-SNR.

Results and Discussion

Figure 2 plots mean GM perfusion-SNR versus T_{ECG} for subject 1. A clear maximum is seen at $T_{ECG} = 500$ ms, and two local minima are seen at $T_{ECG} = 400$ and 700 ms. Table 1 displays mean GM perfusion SNR for both subjects, comparing results from the non-gated experiment to the maximum and minima of the gated experiment. The optimally triggered experiment offers an 18% perfusion-SNR increase over the non-gated experiment for subject 1, and a 31% improvement for subject 2. Figure 3 shows equally windowed perfusion maps from subject 1 for the non-gated experiment, $T_{ECG} = 700$ ms (minimum), and $T_{ECG} = 500$ ms (maximum). A significant SNR improvement is apparent in figure 2c (more yellow and red regions in map).

The presented data support the ideas introduced in the theory section. Specific choices of T_{ECG} can minimize or maximize perfusion-SNR, based on the positioning of the ASL module relative to the flow waveform. An important point is that the waveforms displayed in figure 1b and 1c depict flow in major arteries, and do not consider flow in smaller downstream branches. The true composite profile will be delayed and dispersed in a largely unpredictable manner; therefore, we suggest to empirically determine the ideal T_{ECG} to maximize ASL signal. Practically, however, since the carotid and vertebral artery flow contribute significantly to deliverable tagged spin volume, the delay between the QRS and carotid pulse may be a first-choice estimate for optimizing T_{ECG} .

References: 1) Buxton et al., MRM 40: 383-396 (1998), 2) Wong et al., MRM 39:702 (1998), 3) Hoge, HBM 2004, 4) Ford et al., Phys Meas, 26:477-488 (2005).
Acknowledgements: Siemens Medical Solutions, R.J. Shillman Career Development Award; NCCR; P41RR14075, MIND Institute

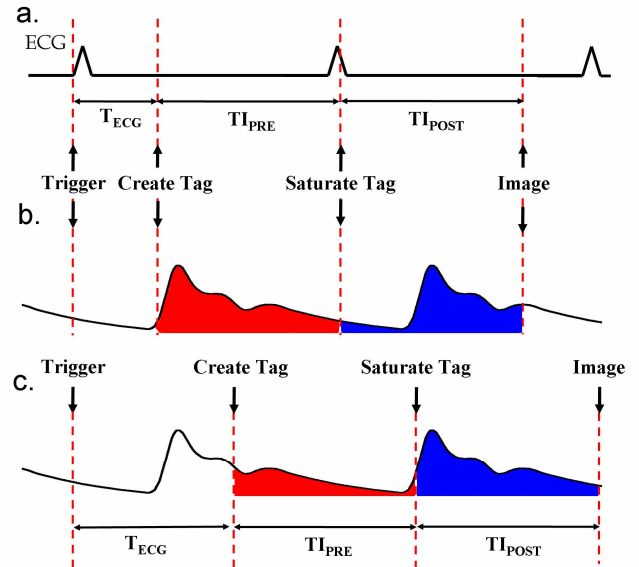
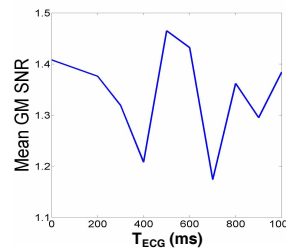


Figure 1. Cardiac gating timing diagram and archetypal carotid flow profiles.



	Mean GM SNR	
	Subject 1	Subject 2
No Trigger	1.218	1.045
Minimum 1	1.208	1.084
Minimum 2	1.174	1.059
Maximum	1.465	1.440

Table 1. Mean gray matter SNR

Figure 2 (left). Mean gray matter SNR versus T_{ECG} for subject 1

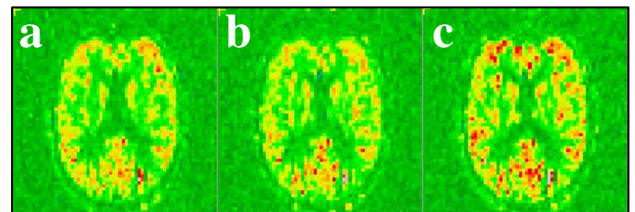


Figure 3. Mean SNR maps from subject 1: a) no trigger, b) $T_{ECG} = 700$ ms (minimum SNR), and c) $T_{ECG} = 500$ ms (maximum SNR)